Although multidrug resistance has commonly been reported in nosocomial *P. aeruginosa*, community-acquired data are less frequently reported. For this reason, epidemiological studies on the prevalence and antimicrobial susceptibility patterns of resistant isolates in different geographical settings, would provide useful information for both empirical treatment of suspected infections and better management of patients.

Our results show that in our setting imipenem is still very active against strains of *P. aeruginosa* in pediatric infections, although other studies have reported higher resistance rates.[3,4] Recent studies have reduced safety concerns over the use of quinolones in pediatric patients. Combined with the good antimicrobial activity shown in this and other studies,[5] this makes them a good empirical choice for community-acquired infections. Local surveillance of antimicrobial activity should be done periodically to guide antimicrobial therapy, especially as carbapenem-resistant *P. aeruginosa* has been reported as an emerging problem in Latin America.[6]

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Unrecognized near-fatal hyperlactatemia in an HIV-infected infant exposed to nucleoside reverse transcriptase inhibitors

Nucleoside analogue reverse transcriptase inhibitors (NRTIs) inhibit mitochondrial DNA (mtDNA) polymerase gamma thereby interfering with mtDNA replication.[1–3] The relative potency of the NRTIs in causing mitochondrial dysfunction is highest for zalcitabine, followed by didanosine, stavudine, and zidovudine. Lamivudine, abacavir, and tenofovir have lower affinity for mtDNA polymerase.[4] In utero and perinatal exposure to NRTIs are known to cause hyperlactatemia (HLA) from mitochondrial toxicity.[5,6]

An HIV-infected mother detected at 30 weeks of pregnancy was treated with lamivudine, stavudine, and nevirapine. A live male baby weighing 2135 g was delivered by elective cesarean section at 38 weeks of gestation with intrapartum intravenous zidovudine cover as per PACTG 076 protocol.[7] He was discharged well with oral zidovudine on the third day.

At three weeks old he developed fever, abdominal distension, diarrhea, and vomiting, which required treatment with intravenous fluids and antibiotics, and was discharged well after 12 days. At five weeks old he returned with diarrhea, poor feeding, dehydration, and acidotic breathing. Arterial blood pH was 7.13, pO_2 128 mmHg, pCO_2 18.7 mmHg, base excess 21.9 mmol/L, and anion gap 24 mmol/L. Serum lactate and ammonia levels were 4.07 mmol/L and 203.6 μmol/L, respectively. Serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), and gamma glutamyltransferase (GGT) levels were 89, 189, and 166 IU/L, respectively. Serum albumin was 28 g/L with prolonged activated partial thromboplastin time (APTT) and increased prothrombin time (PT) ratio. Repeated hemoculture and stool cultures did not yield any pathogen. His urine was screened for inborn errors of metabolism but proved normal. On the second hospital day he developed jerking movements

involving the right arm. A computed tomography scan of the brain was normal and meningitis excluded with cerebrospinal fluid examination. Zidovudine was stopped and his clinical condition improved. HIV infection had been confirmed by HIV-DNA PCR tests carried out earlier. At seven weeks old his serum lactate was 2.69 mmol/L and ammonia was 59.4 mmol/L. The serum ALT and AST levels had normalized to 31 and 27 IU/L, respectively, while the GGT level remained high at 413 IU/L. He was asymptomatic but had moderate global developmental delay.

The clinical features of HLA can be very variable and non-specific, with initial symptoms such as generalized fatigue, muscle weakness, myalgia, gastrointestinal symptoms (nausea, vomiting, diarrhea, abdominal pain, hepatomegaly, anorexia), respiratory involvement (tachypnea and dyspnea), or even neurological symptoms (motor weakness and Guillain–Barre-like syndrome).[8] Hepatic dysfunction may present with encephalopathy with raised liver enzymes but jaundice is unusual.[9] In retrospect the infant had symptomatic HLA at three weeks old, which was unrecognized. Liver dysfunction was evident with elevated liver enzymes and coagulopathy. The seizure was probably due to hepatic encephalopathy complicated by delayed development.

The relative roles of in utero exposure to stavudine and lamivudine and perinatal exposure to zidovudine in causing the severe HLA are unknown; so was the role of HIV infection. Nevertheless, this has posed a challenge in the choice of antiretrovirals for therapy, as the number of available NRTIs less likely to cause HLA is limited.

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Klebsiella ozaenae splenic abscess following odontogenic infection in a girl with sickle cell disease

Hematogenous spread of oral colonizers after dental extraction and dental manipulation is a well recognized problem.[1,2] I report herein a case of splenic abscess and bacteremia due to *Klebsiella ozaenae* following an odontogenic infection.

A 16-year old girl with sickle-beta-thalassemia was hospitalized with a 10-day history of tooth pain and swelling of the cheek. On examination, she was toxic, febrile, and hypotensive with a carious, tender right lower first molar tooth. Purulent exudate was found at the gingival sulcus. Tender submandibular lymphadenopathy, diffuse swelling over the right lower face, and trismus were noted. Four days earlier she had received amoxicillin and aspirin with no relief. Her hemoglobin was 8.8 g/dL and total white cell count was 13.4×10^9/L (94% polymorphs; 11% bands). Radiographic examination revealed pulpitis, periapical infection of the right lower first molar tooth, and an adjacent dentoalveolar abscess. After submitting blood and aspirated pus samples collected from the abscess for culture, treatment with penicillin, chloramphenicol, metronidazole, intravenous fluids, sodium bicarbonate, and analgesics was initiated. On day 3, dental extraction was done and aseptically sampled material from the pulp chamber was submitted for culture. Blood and pus culture specimens yielded a lactose non-fermenting, catalase-positive, facultative Gram-negative bacillus on blood and MacConkey agar. The organism was identified as *Klebsiella ozaenae* and differentiated from other species by its ability to produce a positive methyl red test, *o*-nitrophenyl-β-D-galactopyranoside (ONPG), production of citrate, inability to ferment dulcitol and produce malonate.[2–5] The antimicrobial